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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/875,076	06/06/2001	Chen W. Liaw	AREN-011DIV (11.US9.DIV)	6379
65643 7590 09/04/2007 BOZICEVIC, FIELD & FRANCIS LLP (ARENA PHARMACEUTICALS, INC.) 1900 UNIVERSITY AVENUE SUITE 200 EAST PALO ALTO, CA 94303			EXAMINER LOCKARD, JON MCCLELLAND	
			ART UNIT 1647	PAPER NUMBER
			MAIL DATE 09/04/2007	DELIVERY MODE PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No. 09/875,076	Applicant(s) LIAW ET AL.	
	Examiner Jon M. Lockard	Art Unit 1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 19 June 2007.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 77-101 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 77-101 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 21 March 2003 is/are: a) ☐ accepted or b) ☒ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|---|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date <u>6/19/07, 8/17/07</u> . | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 19 June 2007 has been entered.

Status of Application, Amendments, and/or Claims

2. The Art Unit location of your application in the USPTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Art Unit 1647, Examiner Jon M. Lockard.

3. The Amendment and Response filed 19 June 2007 has been received and entered in full. Claims 77-101 are pending and the subject of this Office Action.

4. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Information Disclosure Statement

5. The information disclosure statements (IDS) submitted on 19 June 2007 and 17 August 2007 have been considered by the examiner.

Maintained and/or New Objections and/or Rejections***Drawings***

6. The drawings filed 21 March 2003 are objected to because Figures 3 and 5 are too dark for the Examiner to reasonably interpret. The drawings filed 21 March 2003 are further objected to because each drawing sheet submitted after the filing date of an application must be labeled in the top margin as either "Replacement Sheet" or "New Sheet" pursuant to 37 CFR 1.121(d). Corrected drawing sheets in compliance with 37 CFR 1.121(d) are required in reply to the Office action to avoid abandonment of the application. Any amended replacement drawing sheet should include all of the figures appearing on the immediate prior version of the sheet, even if only one figure is being amended. The figure or figure number of an amended drawing should not be labeled as "amended." If a drawing figure is to be canceled, the appropriate figure must be removed from the replacement sheet, and where necessary, the remaining figures must be renumbered and appropriate changes made to the brief description of the several views of the drawings for consistency. Additional replacement sheets may be necessary to show the renumbering of the remaining figures. If the changes are not accepted by the Examiner, the applicant will be notified and informed of any required corrective action in the next Office action. The objection to the drawings will not be held in abeyance.

Claim Rejections - 35 USC § 101 and 35 USC § 112, 1st Paragraph

7. 35 U.S.C. 101 reads as follows:

Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 77-101 remain rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific and substantial asserted utility, or a well established utility. Novel biological molecules lack an established utility and must undergo extensive experimentation to determine an appropriate specific and substantial utility. The basis for this rejection is set forth at pg 3-6 of the previous Office Action (mailed 21 March 2003), pg 2-8 of the previous Office Action (mailed 18 March 2005), pg 2-8 of the previous Office Action (mailed 13 October 2005), pg 2-10 of the previous Office Action (mailed 19 May 2006), and pg 2-6 of the previous Office Action (mailed 26 December 2006).

9. The instant application discloses an isolated hARE-2 polypeptide with an amino acid sequence set forth as SEQ ID NO:20 that is encoded by the claimed polynucleotide of SEQ ID NO:19. The specification asserts that the hARE-2 polypeptide encoded by the claimed polynucleotide of the instant invention is believed to be a G-protein coupled receptor (GPCR), having 53% homology with the orphan GPCR GPR27 (See Table A, pg 8). The Specification

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discloses that hARE-2 is expressed in the left and right cerebellum and the substantia nigra (See Table C, pg 27). The specification also teaches that the disclosed human orphan GPCRs may be used for the direct identification of candidate compounds as inverse agonists, agonists, or partial agonists for use as pharmaceutical agents (See pg 15, lines 14-16). There is no well-established utility for a specific hARE-2 nucleic acid or amino acid sequence, and the specification fails to disclose a specific and substantial utility for the claimed invention. The instant application does not disclose a specific biological role for the claimed hARE-2 nucleic acid or the protein encoded by it, or its significance to a particular disease, disorder, or physiological process which one would manipulate for a desired physiological or clinical effect.

10. Applicant's arguments (filed 19 June 2007) as they pertain to the rejections have been fully considered but are not deemed to be persuasive for the following reasons.

11. Applicant prefaces their arguments by asserting at pg 6 of the response that the observation that hARE-2 is selectively expressed in the same cells that die in Parkinson's disease provides at least two utilities for the claimed subject matter. However, it is noted that the specification only discloses that hARE-2 is expressed in the substantia nigra, it does not disclose that hARE-2 is expressed in the same population of cells that die in Parkinson's disease. It is well known in the art that the substantia nigra is composed of a heterogeneous population of neurons, the majority of which are either use GABA as their neurotransmitter (i.e., GABAergic) or use dopamine as their neurotransmitter (i.e., dopaminergic) (See for example Hirsch et al., J. Neural Transm. Suppl. 50:79-88, 1997; cited by Applicant 11/17/06). It is also well established

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in the art that “the generally accepted pathological basis of Parkinson’s disease (PD) is the extensive loss of dopaminergic neurons in the zona compacta of the substantia nigra” (See Leenders et al. Arch. Neurol. 47:1290-1298; cited by Applicant). Therefore, contrary to Applicant’s assertion, the specification does not disclose that hARE-2 is expressed in the same population of cells that dies in Parkinson’s disease, nor has Applicant has not provided any evidence or reference of record to substantiate the allegation that the claimed hARE-2 polynucleotides are expressed in the same cells that die in Parkinson’s disease.

12. Applicant argues at pg 6, 8, and 9 of the response that the selective expression pattern of hARE-2 in substantia nigra cells allows for modulation of the intracellular signaling molecules (cAMP, IP₃ and/or Ca²⁺) selectively in those cells, and asserts that fluctuations in the intracellular levels of these same molecules are correlated with the viability of substantia nigra cells (citing Hulley et al. and Hirsch et al.). Applicant thus argues that hARE-2 can be used to identify compounds that modulate the intracellular levels of cAMP, IP₃, and/or Ca²⁺, resulting in the identification of compounds that stave off or slow the progression of Parkinson’s disease.

13. Applicant’s arguments (filed 19 June 2007) have been fully considered but are not persuasive for the following reasons. While the specification asserts that hARE-2 is a GPCR expressed in the substantia nigra, there is no teaching in the specification as originally filed, or reference of record at the time of the invention, which demonstrate that hARE-2 effects the intracellular level of cAMP, IP₃, and/or Ca²⁺ (via IP₃). It is noted that while Hulley et al. demonstrate that the viability of *dopaminergic* neurons in the substantia nigra are sensitive to the levels of intracellular cAMP, Applicant has failed to demonstrate that putative hARE-2 GPCR is

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associated with effecting changes in intracellular cAMP, or whether activation hARE-2 leads to an increase in cAMP, or a decrease in cAMP. Likewise, while Hulley et al. demonstrate that *dopaminergic* neurons in the substantia nigra are sensitive to the levels of intracellular Ca^{2+} , Applicant has failed to demonstrate that putative hARE-2 GPCR is associated with effecting changes in intracellular Ca^{2+} , or whether activation of hARE-2 leads to an increase or decrease in intracellular Ca^{2+} . Moreover, as noted above, specification does not disclose that hARE-2 is expressed in the same population of cells (i.e., dopaminergic neurons) that dies in Parkinson's disease, nor has Applicant has not provided any evidence or reference of record to substantiate the allegation that the claimed hARE-2 polynucleotides are expressed in the same cells that die in Parkinson's disease.

14. Applicant argues at pg 7 and 9 of the response that the claimed subject matter can be employed to identify compounds that facilitate detection of hARE-2 expressing cells, and thus can facilitate diagnosis and/or monitoring of Parkinson's disease. Applicant argues, for example, that compounds identified by using the claimed composition may be used in radio-imaging methods for the study of Parkinson's disease in a manner similar to the compounds described in Leenders et al. (Arch. Neurol. 47:1290-1298, 1990) and Fischman et al. (Synapse. 29:128-141, 1998). Applicant further argues that hARE-2 is conceptually no different from a marker of other cell types that degenerate during the manifestation of a disease.

15. Applicant's arguments (filed 19 June 2007) have been fully considered but are not persuasive for the following reasons. It is noted that the references submitted by Applicant (Leenders et al. and Fischman et al.) utilized compounds that were selective for dopaminergic

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neurons, which the art recognizes is the population of cells that die in Parkinson's disease. However, as previously discussed *supra*, the specification only discloses that hARE-2 is expressed in the substantia nigra, it does not disclose that hARE-2 is expressed in the same population of cells that die in Parkinson's disease. It is well known in the art that the substantia nigra is composed of a heterogeneous population of neurons, the majority of which are either use GABA as their neurotransmitter (i.e., GABAergic) or use dopamine as their neurotransmitter (i.e., dopaminergic) (See for example Hirsch et al., J. Neural Transm. Suppl. 50:79-88, 1997; cited by Applicant 11/17/06). It is also well established in the art that "the generally accepted pathological basis of Parkinson's disease (PD) is the extensive loss of dopaminergic neurons in the zona compacta of the substantia nigra" (See Leenders et al. Arch. Neurol. 47:1290-1298; cited by Applicant). Therefore, contrary to Applicant's assertion, the specification does not disclose that hARE-2 is expressed in the same population of cells that dies in Parkinson's disease, nor has Applicant has not provided any evidence or reference of record to substantiate the allegation that the claimed hARE-2 polynucleotides are expressed in the same cells that die in Parkinson's disease. Since the specification does not disclose that hARE-2 is expressed in the same population of cells (i.e., dopaminergic neurons) that die in Parkinson's disease, Applicant's assertion that hARE-2 can be employed to identify compounds that facilitate detection of hARE-2-expressing cells and thus be used in the diagnosis and/or monitoring of Parkinson's disease is not substantial.

16. Applicant argues at pg 8 of the response that, contrary to Examiner's argument that hARE-2 lacks utility because it is unclear whether inhibition of hARE-2 is good or bad for

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substantia nigra cells, the rejected claims neither require a hARE-2 inhibitor nor require inhibition of hARE-2 activity.

17. Applicant's arguments (filed 19 June 2007) have been fully considered but are not persuasive for the following reasons. While the Examiner acknowledges that the rejected claims neither require a hARE-2 inhibitor nor require inhibition of hARE-2 activity, the point the previous Examiner was trying to make is that the instant specification leaves it to the skilled artisan to determine whether compounds that inhibit the hARE-2 polypeptide encoded by the claimed polynucleotides could be used in methods of treatment, or whether compounds that potentiate/promote the hARE-2 polypeptide could be used in methods of treatment. Therefore, until one knows what the physiological consequences that the administration of an agonist or antagonist of a receptor protein of the instant invention are going to be, the protein lacks a specific and substantial utility in "currently available form".

18. Applicant argues at pg 8-9 of the response that a correlation between hARE-2 and Parkinson's disease clearly exists because hARE-2 is selectively expressed in the very cells that degenerate to cause Parkinson's disease.

19. Applicant's arguments (filed 19 June 2007) have been fully considered but are not persuasive for the following reasons. As previously discussed *supra*, the specification only discloses that hARE-2 is expressed in the substantia nigra, it does not disclose that hARE-2 is expressed in the same population of cells that die in Parkinson's disease. It is well known in the art that the substantia nigra is composed of a heterogeneous population of neurons, the majority of which are either use GABA as their neurotransmitter (i.e., GABAergic) or use dopamine as

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their neurotransmitter (i.e., dopaminergic) (See for example Hirsch et al., J. Neural Transm. Suppl. 50:79-88, 1997; cited by Applicant 11/17/06). It is also well established in the art that “the generally accepted pathological basis of Parkinson’s disease (PD) is the extensive loss of dopaminergic neurons in the zona compacta of the substantia nigra” (See Leenders et al. Arch. Neurol. 47:1290-1298; cited by Applicant). Therefore, contrary to Applicant’s assertion, the specification does not disclose that hARE-2 is expressed in the same population of cells that dies in Parkinson’s disease, nor has Applicant has not provided any evidence or reference of record to substantiate the allegation that the claimed hARE-2 polynucleotides are expressed in the same cells that die in Parkinson’s disease. Since the specification does not disclose that hARE-2 is expressed in the same population of cells (i.e., dopaminergic neurons) that die in Parkinson’s disease, or that hARE-2 is overexpressed or underexpressed in Parkinson’s disease, one skilled in the art would not be able to predict, with any level of certainty, that hARE-2 is associated with or correlated with Parkinson’s disease.

20. Applicant argues at pg 9 of the response that the facts of this case are completely different to the facts presented in Brenner v. Manson since the substantia nigra-selective expression pattern of hARE-2 provides a very clear path to Parkinson’s disease.

21. Applicant’s arguments (filed 19 June 2007) have been fully considered but are not persuasive for the following reasons. It is noted that Applicant has not provided any evidence or reference of record to substantiate the allegation that the claimed hARE-2 polynucleotide or the protein encoded thereby is involved Parkinson’s disease or any other disease or disorder, or that molecules that interact with it or modulate its activity can be used to treat any disease or

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disorder.

It must be emphasized that arguments of counsel alone cannot take the place of evidence in the record once an examiner has advanced a reasonable basis for questioning the disclosure. See *In re Budnick*, 537 F.2d at 538, 190 USPQ at 424; *In re Schulze*, 346 F.2d 600, 145 USPQ 716 (CCPA 1965); *In re Cole*, 326 F.2d 769, 140 USPQ 230 (CCPA 1964). For example, in a case where the record consisted substantially of arguments and opinions of applicant's attorney, the court indicated that factual affidavits could have provided important evidence on the issue of enablement. See *In re Knowlton*, 500 F.2d at 572, 183 USPQ at 37; *In re Wiseman*, 596 F.2d 1019, 201 USPQ 658 (CCPA 1979).

Until some actual and specific activity or significance can be attributed to the protein identified in the specification as hARE-2 (SEQ ID NO:20) or the polynucleotide encoding it (SEQ ID NO:19), the claimed invention is incomplete. In the absence of a knowledge of the biological significance of this protein, there is no immediately obvious patentable use for it. Furthermore, to employ a protein of the instant invention in the identification of substances which stimulate or inhibit its activity is clearly to use it as the object of further research, which has been determined by the courts to be a utility, which alone, does not support patentability.

22. Whereas one could readily employ the putative hARE-2 protein encoded by the claimed polynucleotides of the instant invention in an assay to identify modulators thereof, the information obtained from such assays would be of little use until one discovers the identity of those physiological processes mediated by that putative hARE-2 protein. Because the instant specification has failed to identify a physiological process which has been shown to be influenced by the activation or inhibition of the putative hARE-2 protein of the instant invention, an artisan would have no way of predicting what effects the administration of that modulator to

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an organism would have. If one cannot predict the effects that the administration of a modulator of the hARE-2 protein of the instant invention is going to have on an organism, then it is unclear as to what practical or real world benefit is derived by the public from the identification of that modulator.

23. It is possible that, after further characterization, the hARE-2 polynucleotides may be found to have a specific and substantial credible utility. This further characterization, however, is part of the act of invention, and until it has been undertaken, Applicant's claimed invention is incomplete. The instant situation is directly analogous to that which was addressed in *Brenner v. Manson*, 148 U.S.P.Q. 689 (Sup. Ct, 1966), in which a novel compound which was structurally analogous to other compounds which were known to possess anti-cancer activity was alleged to be potentially useful as an anti-tumor agent in the absence of evidence supporting this utility. The court expressed the opinion that all chemical compounds are "useful" to the chemical arts when this term is given its broadest interpretation. However, the court held that this broad interpretation was not the intended definition of "useful" as it appears in 35 U.S.C. §101, which requires that an invention must have either an immediately obvious or fully disclosed "real world" utility. The court held that:

"The basic quid pro quo contemplated by the Constitution and the Congress for granting a patent monopoly is the benefit derived by the public from an invention with substantial utility", "[u]nless and until a process is refined and developed to this point-where specific benefit exists in currently available form-there is insufficient justification for permitting an applicant to engross what may prove to be a broad field", and "a patent is not a hunting license", "[i]t is not a reward for the search, but compensation for its successful conclusion."

In the Instant case, the instant specification leaves it to the practitioner to discover the identity of a disease or disorder in which hARE-2 of the instant invention is associated, or which is mutated

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or aberrantly expressed; and then to discover the nature of that aberrant expression (i.e., overexpression or underexpression). The evidence of mere identification as a GPCR based on sequence homology and expression of the mRNA in the cerebellum and substantia nigra is not tantamount to a showing of a role of the claimed polynucleotides or the polypeptides encoded thereby in the diagnosis of Parkinson's disease, or that compounds that modulate its activity are useful in the treatment of Parkinson's disease. Therefore, the claimed polynucleotide or the protein encoded thereby cannot be used in a diagnostic or therapeutic capacity without the need for a substantial inventive contribution. Such additional experimentation, if needed to identify a specific utility for an invention, is precluded by the court. Essentially, Applicant has not provided evidence to demonstrate that the claimed hARE-2 polynucleotide of the instant application is supported by a specific and substantial asserted utility or a well-established utility.

It is noted that M.P.E.P. 2107.01 states:

Deficiencies under the "useful invention" requirement of 35 U.S.C. 101 will arise in one of two forms. The first is where it is not apparent why the invention is "useful." This can occur when an applicant fails to identify any specific and substantial utility for the invention **or fails to disclose enough information about the invention to make its usefulness immediately apparent to those familiar with the technological field of the invention.** Brenner v. Manson, 383 U.S. 519, 148 USPQ 689 (1966); In re Ziegler, 992 F.2d 1197, 26 USPQ2d 1600 (Fed. Cir. 1993). The second type of deficiency arises in the rare instance where an assertion of specific and substantial utility for the invention made by an applicant is not credible (**Emphasis added**).

In the instant case, the instant specification leaves it to the skilled artisan to 1) identify a specific biological role for the encoded hARE-2 polypeptide or its significance to a particular disease, disorder, or physiological process which one would want to manipulate for a desired physiological or clinical effect; and 2) determine whether compounds that inhibit the encoded

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hARE-2 polypeptide could be used in methods of treatment, or whether compounds that potentiate/promote the hARE-2 polypeptide could be used in treatment methods.

24. The Examiner has fully considered all evidence of record and has responded to each substantive element of Applicant's response.

Claim Rejections - 35 USC § 112, 1st Paragraph

25. Claims 77-101 also remain rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a specific and substantial asserted utility or a well established utility for the reasons set forth above, one skilled in the art clearly would not know how to use the claimed invention. The basis for this rejection is set forth at pg 6 of the previous Office Action (mailed 21 March 2003), pg 8 of the previous Office Action (mailed 18 March 2005), pg 8-9 of the previous Office Action (mailed 13 October 2005), pg 10 of the previous Office Action (mailed 19 May 2006), and pg 6 of the previous Office Action (mailed 26 December 2006).

Summary

26. No claim is allowed.

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Advisory Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to **Jon M. Lockard** whose telephone number is **(571) 272-2717**. The examiner can normally be reached on Monday through Friday, 7:00 AM to 4:30 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, **Manjunath N. Rao**, can be reached on **(571) 272-0939**.

The fax number for the organization where this application or proceeding is assigned is **571-273-8300**.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at **866-217-9197** (toll-free).



Jon M. Lockard, Ph.D.
August 28, 2007